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Carbon Dioxide Embolisms During Laparoscopic Surgery

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Abstract

With the advancement of technology over the past few decades, laparoscopic surgery has significantly increased in popularity among many surgical subspecialties including: general surgery, gynecology, and urology. Many procedures that traditionally required open access with large incisions have been replaced with laparoscopic approaches as it offers many advantages such as, faster recovery, less postoperative pain, and shorter hospital stays. Despite its many advantages, it creates numerous anesthesia challenges and considerations when caring for patients undergoing laparoscopic procedures. Among these challenges, are carbon dioxide embolisms. This manuscript involves research and previous case studies that were conducted on carbon dioxide embolisms during laparoscopic surgery. The purpose of this manuscript is to have a solid foundation of research in order to evaluate an event of a suspected carbon dioxide embolism during a laparoscopic, hand-assisted nephrectomy.

Carbon Dioxide Embolisms during Laparoscopic Surgery

Laparoscopic surgery is a commonly used approach for a number of procedures as it is a minimally invasive technique that provides less postoperative pain, faster recovery, and a shorter hospital stay. With its increasing popularity, it is crucial to understand complications that can arise from laparoscopy and how to treat them. Of the complications that can occur, this manuscript will focus on carbon dioxide embolisms and compare and contrast a number of case studies. The case studies discussed will be compared and contrasted to a recent suspected carbon dioxide embolism event that occurred during a laparoscopic, hand-assisted nephrectomy.

A carbon dioxide embolism is defined as an entrapment of carbon dioxide that inadvertently enters the vascular system (Nagelhout & Plaus, 2014). Carbon dioxide embolisms can have devastating effects on the cardiovascular and respiratory system and can quickly lead to cardiac arrest requiring immediate resuscitation. As an anesthesia provider, it is critical to identify a suspected carbon dioxide embolism immediately and to be able to respond in a safe and effective manner.

History of Laparoscopic Surgery

Laparoscopic surgery dates back to 1901, when a German surgeon, George Kelling, attempted the first endoscopic examination of the peritoneal cavity to evaluate the effects of pneumoperitoneum on intraabdominal hemorrhage (Nagelhout & Plaus, 2014). Technological limitations created a high risk of bowel and cautery injuries, as well as, vascular perforation. (Nagelhout & Plaus, 2014). As a result, it did not gain much popularity until the mid 1980s. In 1971, the “open entry Hassan” trocar was developed and soon after in the mid-1980s, the videoscopic imaging was created and the safety of laparoscopic procedures significantly improved (Nagelhout & Plaus, 2014). In 1988, the first videolaparoscopic cholecystectomy was

performed by French surgeon, Philip Mouret, and the laparoscopic approach became much more popular (Nagelhout & Plaus, 2014). Not soon after the first laparoscopic cholecystectomy, surgeons began to explore other uses of the laparoscopic approach including, but are not limited to: hysterectomies, nephrectomies, and prostatectomies.

Establishing the Pneumoperitoneum

Laparoscopic procedures require that a pneumoperitoneum be created to generate space in the peritoneal cavity for the surgeon to be able to see intraabdominal structures and to allow manipulation of instruments with clear visualization (Nagelhout & Plaus, 2014). This decreases the risk of accidental injury while safely performing the surgical procedure. To create an artificial pneumoperitoneum, air or gas is installed into the peritoneal cavity under a controlled pressure. Carbon dioxide is commonly used due to its high diffusion rate and should be installed under a pressure of 15 millimeters of mercury (mm Hg) to reduce the physiologic effects on the different body systems. There are two main techniques commonly used to create an artificial pneumoperitoneum: the closed technique with a Veress needle and the open (Hasson) procedure (Nagelhout & Plaus, 2014). According to Nagelhout & Plaus (2014), extremely thin, obese, or those with known abdominal adhesions are at increased risk of entry-related injuries when the closed technique at the umbilical entry point is used compared to the open (Hasson) or left upper-quadrant (Palmer's point) entry technique.

The closed technique is a commonly used technique and involves using the Veress needle, a spring-loaded needle. The Veress needle is used to penetrate the abdominal wall either below or into the umbilicus. An intraabdominal pressure of 10 mmHg or less has been shown to be a reliable measurement of correct placement (Nagelhout & Plaus, 2014). Once placement is confirmed, carbon dioxide is instilled to create the pneumoperitoneum by increasing the

intraabdominal pressure, raising the abdominal wall off organ structures to create space for instruments. After the pneumoperitoneum is established, a trocar is inserted either blindly or under direct vision to allow the insertion of instruments into the abdominal cavity (Nagelhout & Plaus, 2014). This technique is commonly used by surgeons today.

H. M. Hasson developed the open technique in 1970 and it involves making a 2.5-millimeter (mm) incision vertically at the lower border of the umbilicus into the subcutaneous tissue and underlying fascia (Nagelhout & Plaus, 2014). After the incision is made, the surgeon is able to directly detach the abdominal wall from the underlying tissues and once the abdominal cavity has been opened, a trocar is placed with direct vision and sutured in place. Once the trocar is in place, carbon dioxide can be instilled through a side port in the Hasson trocar to establish the pneumoperitoneum. This technique reduces the risk of inadvertent injury to the bowel and vasculature; however, it is not as commonly used (Nagelhout & Plaus, 2014).

Characteristics of Carbon Dioxide

Carbon dioxide is the gas of choice when insufflating the abdomen to create a pneumoperitoneum of laparoscopic surgery. There are numerous reasons why it is superior to using air. Carbon dioxide is colorless, non-toxic, non-flammable, and highly soluble in blood (Hall, 2016). It is also inexpensive and readily available (Park, Kwon, & Kim, 2012). Its characteristics make one of the safest gases to use during laparoscopic surgery and has the lowest risk of gas embolism due to its high diffusion rate (Park et al., 2012). If an embolism occurs that creates significant physiologic compromise, the carbon dioxide embolism, by principle, should eventually dissolved into the blood. However, carbon dioxide does have negative side effects including: hypercapnia, metabolic acidosis, cardiorespiratory compromise, and increased postoperative pain (Park et al., 2012). Due to its associated side effects that can occur, other

gases are being investigated, such as, argon. Below is a table from Park et al. (2012, p. 461) representing the blood/gas solubility of various gases:

Gas	Solubility (blood/gas)*
Oxygen	0.024 [†]
Nitrogen	0.013
Nitrous oxide	0.45
Argon	0.026
Carbon dioxide	0.60
Helium	0.008

*Solubility in mL gas/mL solvent with 100% gas and at 17°C.

[†]Not including that bound to hemoglobin.

Park et al. (2012) describes how the clinical effects of carbon dioxide embolisms depend on the ratio of the volume of carbon dioxide entered into circulation to the amount of carbon dioxide that is removed from the circulation. As cited in Park et al. (2012), Dion et al, (1995) conducted a study that found that a mean of 300 milliliters (mL) of carbon dioxide was necessary to cause death in a 35-kilogram (kg) dog, which translated to requiring over 600 mL to cause death in a 70 kg human. By theory, it seems that it would take a significant amount of inadvertent carbon dioxide infusion into circulation to cause fatality.

Incidence

According to Nagelhout & Plaus (2014), animal and human studies using a transesophageal echocardiography (TEE) show that the incidence of carbon dioxide embolism during laparoscopic procedures occurs 65-100% of the time and that the majority of these embolisms show some respiratory and cardiovascular compromise that were usually resolved spontaneously. However, a significant carbon dioxide embolism resulting in detrimental outcomes are rare (Naghelout & Plaus, 2014). The incidence of a significant carbon dioxide

embolism occurs as low as 0.001% to 0.59% of the time, but when it does occur, the mortality rate is as high as 28.5% (Nagelhout & Plaus, 2014). Carbon dioxide embolisms can occur at any time during laparoscopic procedures if there are intravascular openings that have a lower pressure than the intraabdominal pressure (Nagelhout & Plaus, 2014). It can also occur if the Veress needle is incorrectly placed and carbon dioxide is inadvertently instilled into an intraabdominal vessel (Nagelhout & Plaus, 2014). In a meta-analysis with closed-entry laparoscopies, carbon dioxide emboli occurred 0.001% of the time and similar to Nagelhout & Plaus (2014), a mortality rate was reported to be 28% (Park et al., 2012). Smith (2011) reported an incidence of carbon dioxide embolisms as 0.0014% to 0.6% and an associated mortality rate of 28%. As stated above, a consistent incidence of carbon dioxide embolisms occurs roughly 0.1% to 0.6% in laparoscopic procedures with a mortality rate of approximately 28%. Smith (2012) found that the incidence of carbon dioxide embolisms was higher in laparoscopic gynecological procedures associated with hysteroscopy than any other procedures.

Pathophysiology

The pathophysiology of a carbon dioxide embolism is similar to that of any type of gas embolism. A large amount of carbon dioxide must enter circulation through an artery, vein, or solid organ such as, the liver (Nagelhout & Plaus, 2014). The carbon dioxide embolism travels up the inferior vena cava, through the right atrium and right ventricle, and lodges itself into the pulmonary artery or pulmonary circulation (Nagelhout & Plaus, 2014). This mechanism is also referred to as a “gas lock” (Park et al., 2012). This disruption causes an increase in right ventricular workload leading to right ventricular failure, increased pulmonary artery pressure that can lead to pulmonary arterial hypertension, and decreased pulmonary venous return (Nagelhout & Plaus, 2014). Left untreated, the decrease in venous return leads to decreased left ventricular

preload, decreased cardiac output, severe hypotension, asystole, and cardiovascular collapse (Nagelhout & Plaus, 2014). Carbon dioxide embolisms produce similar, but less significant effects than air because carbon dioxide has the ability to diffuse into the blood and alleviate the physiologic response to that embolism (Park et al., 2012). In addition, carbon dioxide embolisms, in comparison to air embolisms, do not cause bronchoconstriction or decreased pulmonary compliance (Park et al., 2012).

Signs and Symptoms

In a patient under general anesthesia, signs and symptoms include: a sudden decrease in end-tidal carbon dioxide (ETCO₂), a sudden rise in end-tidal nitrogen, increased pulmonary artery pressures, marked hypotension, dysrhythmias, hypoxia, cyanosis, pulmonary edema, and a “mill” wheel murmur that can be auscultated with a precordial or esophageal stethoscope (Nagelhout & Plaus, 2014). The auscultation of the classic mill wheel murmur means that two milliliter per kilogram (mL/kg) or more of carbon dioxide is entrained into the right side of the heart (Nagelhout & Plaus, 2014). When this much carbon dioxide is entrained in the heart, there will be significant hemodynamic effects that occur such as, tachycardia, hypotension, cardiac dysrhythmias, cyanosis, and electrocardiogram (ECG) changes indicative of right sided heart strain (Nagelhout & Plaus, 2014). On the contrary, Smith (2011) states that the ETCO₂ reading has a sudden increase and then rapid decrease that may progress to a complete loss in ETCO₂ waveform tracing. The sudden drop in ETCO₂ is caused by a reduction in pulmonary blood flow resulting in significant decreased perfusion (Smith, 2011). Park et al. (2012) states that ETCO₂ can either increase or decrease during a carbon dioxide embolism; however, most reports stated that there was a sudden decrease rather than increase in ETCO₂. As cited in Park et al. (2012), Diakun (1991) describes that a rise in ETCO₂ is from carbon dioxide that has entered circulation

and is being readily dissolved into the blood; whereas, a sudden drop in ETCO₂ is due to an abrupt obstruction from an embolism in the pulmonary vasculature. Carbon dioxide embolism causes a gas exchange abnormality that physiologically decreases the partial pressure of oxygen and significantly decreases perfusion of oxygen and carbon dioxide across the pulmonary alveolar membranes (Nagelhout & Plaus, 2014).

Diagnosis

Diagnosis of carbon dioxide embolisms are dependent on the rapid recognition of the physiologic signs and symptoms of embolism and/or direct visualization of an embolism in the right side of the heart and/or in the pulmonary vasculature, including the pulmonary artery (Nagelhout & Plaus, 2014). Transesophageal echocardiography is known to be the most sensitive diagnostic tool used in detecting carbon dioxide embolisms and can detect an embolism as small as 0.1 mL/kg (Park et al., 2012). However, TEE it is not a standard monitoring tool in the operating room due because it is complex, expensive, and cumbersome in nature (Park et al., 2012). While in use, the provider must be holding it and have constant attention on the device making it extremely hard to maintain other aspects of anesthesia care such as, fluid administration, medication administration, and airway management. In reality, it would require a second anesthesia provider to remain in the room at all times. TEE is not commonly readily available in the room and would require someone to either leave the operating room to obtain it or require someone outside the operating room to be notified to bring it in, which both require time and resources. Since carbon dioxide is so highly soluble and is rapidly diffused into the bloodstream, it can be difficult to capture the direct visualization of the embolism since it can take several minutes to obtain the TEE equipment.

Park et al. (2012) describes the use of a transesophageal doppler as an easier and cheaper technique to use routinely and that it is “nearly” as sensitive as a transesophageal echocardiography (p. 462). In a study with pigs undergoing laparoscopic cholecystectomy, it was found that the transesophageal doppler demonstrated 100% sensitivity in detecting 0.1 mL/kg emboli (Park et al., 2012). It is also stated that it provides earlier carbon dioxide emboli detection earlier than ETCO₂ monitoring (Park et al., 2012).

The precordial doppler is a noninvasive device that is placed in the parasternal area at the fourth intercostal space over the right atrium (Park et al., 2012). It was advertised to be a highly sensitive tool to detect gas emboli, however; studies have shown that it not as sensitive as the TEE or transesophageal doppler (Park et al., 2012). Although this device is easy to place and is less invasive than other devices, it produces false negatives and can fail to detect the presence of carbon dioxide emboli (Park et al., 2012).

End-tidal carbon dioxide monitoring is a noninvasive technique to detect carbon dioxide emboli (Park et al., 2012). However, a carbon dioxide embolus can either cause an increase or decrease in capnography (Park et al., 2012). An increase in ETCO₂ is due to carbon dioxide rapidly dissolving into the bloodstream causing a higher concentration of carbon dioxide present in the blood. Park et al. (2012) states that there are several reports of a sudden rise in ETCO₂ with a 3.8 to 4.2% increase for those diagnosed with carbon dioxide embolus based on the sudden onset of systolic and diastolic murmurs during laparoscopic surgery (p. 463). A rapid decrease in ETCO₂ occurs when a carbon dioxide embolus is so large that it halts perfusion by causing an obstruction in the pulmonary vasculature leading to increased dead space (Park et al., 2012). Despite several reports of a sudden rise in end-tidal carbon dioxide, most reports show a sudden decrease in ETCO₂ (Park et al., 2012).

Precordial or esophageal stethoscopes are useful in detecting the classic mill-wheel murmur. A mill-wheel murmur is described as “a loud, harsh, churning, tickling, splashing metallic sound, which are usually noted concomitantly with cardiovascular collapse” (Park et al., 2012, p. 463). However, it is important to understand that the presence or sound of the murmur is inconsistent and is not always present during a carbon dioxide embolus (Park et al., 2012). A review of seven episodes of carbon dioxide embolism during laparoscopic surgery summarize that less than half of the patients had a reported murmur (Park et al., 2012). Brundin et al. (1989), stated that the “typical metallic heart sounds were noted during hysteroscopy in 10% of cases” (as cited in Park et al., 2012, p. 463).

Pulmonary artery monitoring is the most invasive technique for detecting a carbon dioxide embolus. Park et al. (2012) describes a study in dogs, where fifteen mL boluses of carbon dioxide were injected into each dog and a TEE was used to detect gas bubbles. Over 100 mLs of carbon dioxide were required to show a rise in pulmonary artery pressure (Park et al., 2012). It does not appear that pulmonary artery pressure is as sensitive or as immediate as other monitoring techniques described above.

Treatment

Rapid recognition is essential in the prompt treatment of carbon dioxide embolism. Failure to recognize and treat a carbon dioxide embolism can quickly result in a fatal outcome, such as, cardiac arrest. Once a provider recognizes the signs that a carbon dioxide embolism is suspected, he or she must first, inform the surgeon to discontinue the carbon dioxide insufflation and to let down the pneumoperitoneum (Park et al., 2012). In addition, Nagelhout and Plaus (2014) mentions to direct the surgeon to flood the surgical field with saline to prevent further

carbon dioxide from entering the vasculature. Help should be called immediately into the room, as well as, a code cart.

Second, give 100% oxygen and discontinue nitrous oxide if being used (Park et al., 2012). While applying 100% oxygen, the respiratory rate and tidal volume should be increased, as well as, increasing positive end-expiratory pressure (PEEP) to decrease the carbon dioxide entrainment (Smith, 2011). Nitrous oxide does not expand carbon dioxide embolus, but does with air; therefore, it is important to stop immediately in the circumstance that it is an air embolus instead of a carbon dioxide embolus (Smith, 2011).

Third, place the patient in left lateral decubitus with steep Trendelenburg or also termed the “Durant’s position” (Smith, 2011). Durant’s position keeps the carbon dioxide trapped in apex of the right atrium and stops the embolus from traveling into the pulmonary outflow tract (Smith, 2011).

Fourth, increase the administration of intravenous fluid administration to adequately hydrate the patient to reduce further entry of gas, which increases the central venous pressures in the heart and supports blood pressure management (Park et al., 2012). Consider placing a second intravenous catheter to enhance the administration of intravenous fluids.

Fifth, consider the insertion of a central venous catheter to allow aspiration of the carbon dioxide from the right atrium (Park et al., 2012). A central venous catheter would allow for a quicker diagnosis by positively aspirating gas bubbles and would significantly improve the hemodynamic stability of the patient by relieving the gas lock in the right side of the heart (Park et al., 2012). However, the insertion of a central venous catheter can be difficult when rapidly resuscitating a patient.

Sixth, resuscitation efforts should be initiated as fast as possible, including: administration of vasopressors and inotropes to maintain an adequate cardiac output and initiation of cardiopulmonary resuscitation if cardiac arrest occurs (Park et al., 2012). Vital signs should be assessed continuously. If cardiac output cannot be adequately maintained with the use of medications and fluids, consider the insertion of an intra-aortic balloon pump (Park et al., 2012). Prostaglandin analogues or phosphodiesterase inhibitors should be considered in the setting of severe pulmonary hypertension (Park et al., 2012).

Seventh, if the patient is successfully resuscitated, the use of hyperbaric oxygen therapy should be considered in the setting of neurological deficits caused by cerebral carbon dioxide emboli (Park et al., 2012). Hyperbaric oxygen therapy reduces the bubble size by one third, decreases blood flow, and limits the significant effects of the gas-blood interface (Park et al., 2012). Other benefits include: decreased intracranial pressure and increased tissue oxygenation due to the diffusion of the emboli (Park et al., 2012). However, hyperbaric oxygen therapy is more useful in air emboli compared to carbon dioxide emboli because of the high diffusion rate of carbon dioxide (Park et al., 2012). By the time a patient is stable enough to enter a hyperbaric oxygen chamber, the carbon dioxide embolus would most likely already be absorbed. Below is a list of interventions for suspected carbon dioxide embolism in chronological order (Park et al., 2012, p. 464):

Inform the surgeon
Prevent further gas entry
Discontinue carbon dioxide insufflation
Discontinue nitrous oxide and ventilate with 100% oxygen
Left lateral decubitus position
Adequate hydration
Central venous catheterization: aspiration of carbon dioxide
Resuscitation
Fluid administration
Drugs:
Inotropes
Vasopressors
Vasodilator specific to pulmonary circulation
Cardiopulmonary resuscitation
Hyperbaric oxygen therapy

Prevention

There are a number of measures that can be taken to help reduce the risk of carbon dioxide embolism. Prevention measures must take place on both the surgical side, as well as, the anesthesia side of the operating room.

On the surgical side, the surgeon must correctly place the Veress needle and confirm placement before instilling carbon dioxide into the intraabdominal space to avoid a massive transfusion of carbon dioxide either intravenously or into a solid organ (Park et al., 2012). Correct placement can be accomplished by aspirating before insufflation and/or testing the inflation with a few milliliters of carbon dioxide (Park et al., 2012). Implementing low insufflation pressures less than 15 mmHg during laparoscopic surgery plays a key role in preventing carbon dioxide emboli (Park et al., 2012). As an anesthesia provider, it is also our role to ensure that the surgical team is adhering to low insufflation pressures.

For the role of the anesthesia provider, there are several measures that can be taken to decrease the risk of hemodynamic collapse if a carbon dioxide embolus were to enter circulation. Firstly, increase the central venous pressure by administering an adequate amount of intravenous fluids helps reduce the risk of a carbon dioxide embolus from causing entrainment in the right side of the heart. Ways to ensure adequate hydration without the use of a central venous pressure monitoring system include: calculating fluid deficit and maintenance rates based on weight, assessing fluid status of the patient based on dry mucus membranes, and the presence of respiratory variation on the oxygen saturation waveform. Placing the patient in steep Trendelenburg would force gas bubbles or emboli that are present to rise to the apex of the right side of the heart and prevent it from advancing to the pulmonary circulation (Park et al., 2012). The use of PEEP may reduce the pressure gradient between the vessel opening and the heart; therefore, decreasing the risk of carbon dioxide entry into the vasculature (Park et al., 2012). Nasrallah and Souki (2018) summarizes that the best measures to take in preventing carbon dioxide emboli as the anesthesia provider is to ensure:

1. Low insufflation pressure less than 15 mmHg
2. Hydration
3. PEEP
4. Trendelenburg positioning

Literature Review

Randomized control trial.

A clinical study, conducted by Xu and Zhang (2013), examined the use of transthoracic echocardiography combined with ETCO₂ monitoring in preventing venous air embolisms during hysteroscopy procedures. Xu and Zhang (2013) enrolled 300 cases undergoing surgery involving

hysteroscopy under general anesthesia from August 2010 to August 2012. Xu and Zhang (2013) evaluated, using a transthoracic echocardiography and ETCO₂ monitor, the time of when a venous air embolism appeared in the right atrium, the extent of air embolism (graded zero to four), changes in ETCO₂, peripheral oxygenation saturation, blood pressure, and the amount of fluid intravasation and blood loss. Venous air embolisms graded three or above with a decrease in ETCO₂ greater than or equal to five mmHg were placed in the intervention group (Xu & Zhang, 2013). Of the 300 cases, twenty-four of those cases were placed in the intervention group (Xu & Zhang, 2013). Measures taken in the intervention group included: cessation of operation, position change to left side with right shoulder raised, oxygen inhalation, and ten milligrams (mg) of dexamethasone given through an intravenous drip (Xu & Zhang, 2013). The criteria for the control group was a grade three or above venous air embolism and that there was a decrease in ETCO₂ that was less than five mmHg (Xu & Zhang, 2013). The control group consisted of five cases.

Xu and Zhang (2013) found that the occurrence of venous air embolism occurred in thirty-four of the patients. All thirty-four patients had evidence of gas embolism in the inferior vena cava, right atrium, and right ventricular chamber (Xu & Zhang, 2013). Twenty-nine of those patients had evidence of continuous gas emboli (Xu & Zhang, 2013). Thirty-two cases had a reduction in ETCO₂ greater than two mmHg, fourteen cases with an oxygen saturation of less than 95%, and four cases with a reduction in blood pressure of greater than or equal to 20% of baseline (Xu & Zhang, 2013). To conclude Xu and Zhang's (2013) study, it was found that the decrease in ETCO₂ was positively correlated to the extent of venous air embolism, ETCO₂ monitoring was more sensitive than oxygen saturation and blood pressure measurements, and

that the amount of intravasation of distention of fluid and blood loss positively correlated to the extent of venous air embolism.

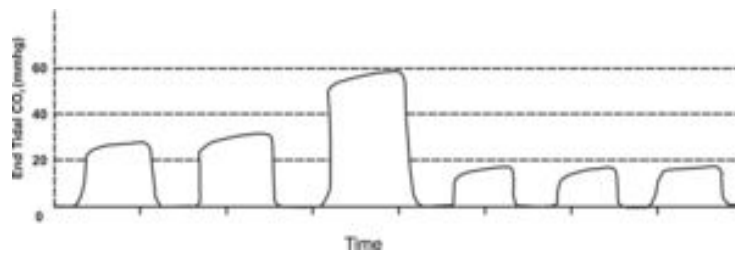
Case studies.

A total of twelve case studies are included in this literature review. A number of similarities and differences are found throughout and will be discussed later in more detail. The case studies are organized by the timing of when the suspected carbon dioxide embolism occurred.

Suspected carbon dioxide embolic events that occurred during dissection

This case study involves a 23-year-old female undergoing bilateral adrenalectomy via the retroperitoneoscopic approach undergoing general anesthesia (Abraham, Jose, & Paul, 2018). The patient underwent induction and intubation with an endotracheal tube uneventfully. After successful intubation, anesthesia was maintained at about 0.8-0.9% minimum alveolar concentration of isoflurane in a 50% oxygen and 50% air (Abraham et al., 2018). Nitrous oxide was not used during this case. The ventilator settings were not listed and the use PEEP is not stated. Intravenous fluids were maintained between 6-8 mL/kg per hour. A target ETCO₂ of 35-45 mmHg was maintained throughout surgery, with the exception of ten minutes when a carbon dioxide embolism was suspected (Abraham et al., 2018). An internal jugular central venous catheter was inserted without complication (Abraham et al., 2018). The patient was placed in the prone position for the procedure. Carbon dioxide insufflation was carried out at 200 mL per minute and a retroperitoneal pressure was maintained at fifteen mmHg (Abraham et al., 2018). The left adrenal gland was removed without complication and the patient remained hemodynamically stable throughout dissection (Abraham et al., 2018). The surgeon then clamped the right adrenal vein about ninety minutes into the total surgical time and a sudden rise

in the ETCO₂ monitor from 35 to 58 mmHg was noted followed by a sudden drop to eighteen mmHg (Abraham et al., 2018). At the same time, ST segment depression occurred changing from 0.0 to -3.5 millimeters (mm) with a decrease in blood pressure from 120/70 mmHg to 60/30 mmHg without any changes in heart rate (Abraham et al., 2018). A carbon dioxide embolism was quickly suspected and treatment included: informing surgeon, 100% oxygen, and the patient repositioned to the Durant's position (Abraham et al., 2018). Hypotension was aggressively treated with a 500 mL bolus of crystalloid and a total of twenty-five mg of ephedrine. The central venous catheter was simultaneously aspirated and yielded about four mL of gas (Abraham et al., 2018). Ten minutes later, the patient was hemodynamically stable with ST segments, blood pressure, and ETCO₂ at baseline (Abraham et al., 2018). Below is a snapshot of the capnography waveform from when the ETCO₂ briefly increased followed by a significant decrease (Abraham et al., 2018):



Abraham et al. (2018) explains that the suspected carbon dioxide embolism occurred due to an opening in the inferior vena cava inadvertently made during the clamping of the right adrenal vein, which occurred about ninety minutes into the surgery. The right adrenal vein is located in close proximity to the inferior vena cava.

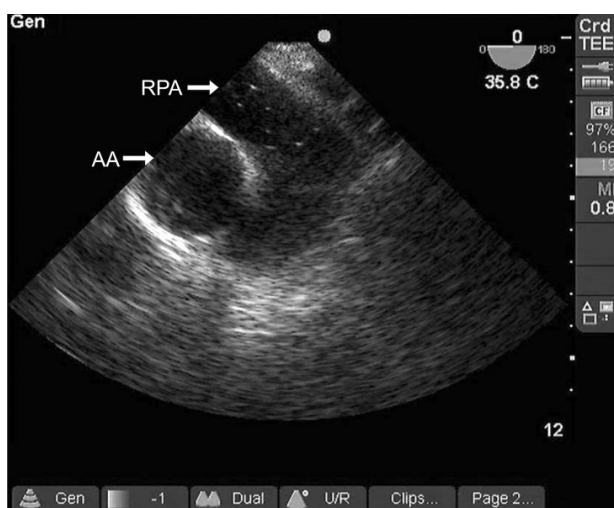
A case study conducted by Aggarwal, Gupta, and Prasad (2016) involves a 25-year-old female undergoing a laparoscopic cholecystectomy. The patient underwent induction and intubation with an endotracheal tube without any complications and maintenance of anesthesia included 60% oxygen and 40% air mixture with sevoflurane at full minimum alveolar

concentration, as well as, a remifentanyl infusion running between 100-400 micrograms per hour (Aggarwal, 2016). The ventilator settings included: intermittent positive pressure ventilation, tidal volume of 400 mL, respiratory rate of 18 breaths per minutes, and PEEP of 6 centimeters of water (cm H₂O) (Aggarwal, 2018). The patient was in the supine position for the length of the procedure. The amount of intravenous fluids is unknown. Creation of the pneumoperitoneum and insertion of the trocars were uneventful and the target insufflation pressure was set to 15 cm H₂O (Aggarwal, 2018). The surgeon noticed “profuse oozing from the gallbladder bed” after clipping the cystic duct and artery (Aggarwal, 2018, p. 002). Within a minute, the ETCO₂ suddenly decreased from 37 to 7 mmHg accompanied by cyanosis, oxygen saturation of 50%, heart rate reduction from 96 to 40 beats per minute, a faint brachial pulse on palpation, a systolic blood pressure of 60 mmHg, and a diastolic blood pressure that was unreadable (Aggarwal et al., 2018). The anesthesia provider immediately called for extra help, turned oxygen up to 100%, switched the ventilator to manual mode, notified surgeons to release pneumoperitoneum, infused intravenous fluids rapidly, repositioned patient to the Durant’s position (Aggarwal et al., 2018). Cardiac arrest occurred after repositioning the patient to the Durant’s position and patient was immediately placed back in supine to initiate compressions (Aggarwal et al., 2018). Epinephrine 1 mg was given and a right internal jugular central venous line was inserted in the midst of cardiopulmonary resuscitation. Over 25 mL of gas was aspirated from all three ports (Aggarwal et al., 2018). Return of spontaneous circulation occurred thirteen minutes into resuscitative efforts and the patient gradually returned to baseline about 15 minutes after return of pulse (Aggarwal et al., 2018). In reflection, Aggarwal et al. (2018), states that the central venous catheter insertion was most critical step in resuscitating the patient from the carbon dioxide embolism because it allowed a large amount of gas to be aspirated from its ports. Currently, the

recommended guidelines for treatment of a carbon dioxide embolism do not state or recommend the emergent insertion of a central venous catheter (Aggarwal et al., 2018).

Kim, Jung, and Shin (2012) describe a case, in which, a suspected carbon dioxide embolism occurred during a laparoscopic low anterior resection and hepatic tumorectomy on a 40-year-old female. Induction and intubation with an endotracheal tube was uneventful and the patient was placed on 50% oxygen and 50% nitrous oxide with sevoflurane between 1.5 to 2% (Kim et al., 2012). The ventilator settings were initially set on volume control ventilation with a tidal volume of 500 mL, respiratory rate of ten; however, PEEP was not stated (Kim et al., 2012). An arterial line was placed in the right radial artery and a central venous catheter was inserted into the right internal jugular vein (Kim, et al., 2012). The patient was initially placed in Trendelenburg for the lower anterior resection and was later placed in left lateral position with reverse Trendelenburg for the hepatic tumorectomy (Kim et a., 2012). After a position change to left lateral and reverse Trendelenburg, the ETCO₂ increased to 48 mmHg (Kim et al., 2012). The target ETCO₂ range was 30 to 35 mmHg (Kim et al., 2012). To combat hypercarbia, the ventilator settings were adjusted to a tidal volume of 400 mL and a respiratory rate of 18 breaths per minute; the ETCO₂ decreased to 32 mmHg (Kim et al., 2012). The insufflation pressure was set at 15 mmHg and the surgery was uneventful until the hepatic vein was unintentionally disrupted during the dissection of liver bed (Kim et al., 2012). During this time, the systolic blood pressure decreased from 110 to 62 mmHg and an arterial blood gas was drawn (Kim et al., 2012). The blood pressure was quickly treated with 10 mg of ephedrine and the intravenous fluid administration was increased (Kim et al., 2012). The results of the arterial blood gas were as follows: pH 6.9, partial pressure of carbon dioxide (PaCO₂) 69 mmHg, bicarbonate 24 milliequivalents per liter (mEq/L), partial pressure of oxygen (PaO₂) 259 mmHg, hemoglobin

9.4 grams per deciliter (g/dL), and hematocrit 29% (Kim et al., 2012). Shortly after the decrease in systolic blood pressure, the ETCO₂ waveform suddenly decreased; however, the values of the capnography were not specifically stated (Kim et al., 2012). The decrease in ETCO₂ was accompanied by a reduction in heart rate ranging from 30 to 40 beats per minutes, and an absence of pulsatile activity on the arterial line waveform. Cardiopulmonary resuscitation was initiated with compressions, 1 mg epinephrine boluses administered two separate times, and 0.5 mg atropine (Kim et al., 2012). At the same time, oxygen was increased to 100% and the patient was placed in Trendelenburg (Kim et al., 2012). Two minutes after the initiation of cardiopulmonary resuscitation, a pulsatile arterial waveform appeared on the monitor (Kim et al., 2012). A TEE was performed that showed air bubbles that were present in the right pulmonary artery (RPA) that can be seen in the picture below (Kim et al., 2012, p.470):



AA: ascending aorta; RPA: right pulmonary artery

It took approximately 15 minutes for the patient to return to a stable condition and a second arterial blood gas was drawn. The results were as follows: pH 7.28, PaCO₂ 34 mmHg, HCO₃ 15.4 mEq/L, PaO₂ 499 mmHg, hemoglobin 6.8 g/dL, and hematocrit 22% (Kim et al., 2012). Based on the low hemoglobin value, 2 units of packed red blood cells were transfused. The

patient was transferred to the intensive care unit, regained consciousness within 20 minutes of transfer, and extubated two hours later without any neurological deficits (Kim et al., 2012)

Shen, Ji, Yang, Wang, and Yang (2011) conducted a case study on a 72-year-old male, who underwent a laparoscopic right nephrectomy and experienced a carbon dioxide embolism. Endotracheal intubation was successful and mechanical ventilation was adjusted to maintain a goal of ETCO₂ between 33 and 38 mmHg (Shen et al., 2011). The anesthesia and oxygen mixture were not specified and the ventilator setting were not explicitly stated. An arterial blood gas was drawn after induction and results were: pH 7.44, PaCO₂ 38 mmHg, and PaO₂ 517 mmHg (Shen et al., 2011). The patient was placed in the Durant's position for the length of the procedure and a pneumoperitoneum was created and maintained at 15 mmHg (Shen et al., 2011). However, during the procedure, the inferior vena cava was inadvertently incised (Shen et al., 2011). During this time, about 30 mL of blood loss occurred (Shen et al., 2011). The central venous pressure gradually increased from 7 to 21 mmHg in the next 10 minutes after the inferior vena cava incision (Shen et al., 2011). Vital signs remained stable throughout the event, but a millwheel murmur was audible (Shen et al., 2011). The central venous catheter was aspirated several times and yielded about 80 mL of gas (Shen et al., 2011). The central venous catheter was then advanced into the right atrium, where another 15 mL of gas was aspirated (Shen et al., 2011). A carbon dioxide embolism was confirmed based on the aspiration of a total of 95 mL of gas and the presence of the classic millwheel murmur (Shen et al., 2011). At the same time, the incision in the inferior vena cava was clipped by the surgeon and the pneumoperitoneum was released (Shen et al., 2011). An arterial blood gas was drawn at the time of the event and results include: pH 7.34, PaCO₂ 46 mmHg, PaO₂ 470 mmHg. About 20 minutes later, the millwheel murmur was no longer audible and the laparoscopic procedure proceeded with a

pneumoperitoneum that was maintained between 10 and 12 mmHg (Shen et al., 2011). The patient fully recovered without any deficits or complications (Shen et al., 2011).

Vora, Shah, Parikh, and Modi (2013) conducted a case study of a 35-year-old female undergoing a transperitoneal laparoscopic Boari's ureteric preimplantation, who survived a suspected carbon dioxide embolism during the dissection of the ureters. The authors did not explicitly describe the intubation or what the patient received for anesthesia gas, but did state that the patient received a "balanced general anesthesia" (Vora et al., 2013, p. 190). The patient was placed in the supine position with a 20-degree Trendelenburg tilt (Vora et al., 2013). A pneumoperitoneum was created and maintained at an intraabdominal pressure of 14 mmHg (Vora et al., 2013). About 2 hours into the surgery, during the ureteral dissection, the ETCO₂ suddenly dropped to 8 mmHg, accompanied by a heart rate of 130 beats per minute with frequent premature ventricular contractions (PVCs) (Vora et al., 2013). At this time, the blood pressure and oxygen saturation remained stable (Vora et al., 2013). An endotracheal blockage and surgical bleeding was ruled out. A carbon dioxide embolism was suspected (Vora et al., 2013). The authors point out that the infusion tubing was patent and no drugs were being administered at this time to rule out suspected air embolisms from intravenous infusions (Vora et al., 2013). The pneumoperitoneum was released and the patient was placed in a 45-degree Trendelenburg tilt. An 80 mg lidocaine bolus was given to treat PVCs and the heart rhythm converted to sinus bradycardia at 42 beats per minute that required a 0.6 mg atropine bolus (Vora et al., 2013). The oxygen saturation, at this time, decreased from 98% to 77% accompanied by the appearance of cyanosis with a non-recordable blood pressure and heart rhythm (Vora et al., 2013). The ECG showed a complete heart block, which suggested pulseless electrical activity in the presence of a non-recordable blood pressure (Vora et al., 2013). Treatment included: one liter (L) of crystalloid

quickly transfused, manual ventilation with 100% oxygen, continuous boluses of atropine (a total of 1.8mg were given), cardiopulmonary resuscitation, and 1 mg of epinephrine (Vora et al., 2013). With little response to resuscitative efforts, dopamine and norepinephrine infusions were initiated and a central venous catheter was inserted through the right internal jugular vein (Vora et al., 2013). Attempted aspiration of air or carbon dioxide through the central venous catheter was unsuccessful and a central venous pressure read 15 mmHg (Vora et al., 2013). After 20 minutes of cardiopulmonary resuscitation, the patient recovered with stable vital signs and a heart rhythm that showed sinus rhythm with evidence of right heart strain (Vora et al., 2013). The patient was transferred to the intensive care and regained consciousness after 2 hours without any signs of neurological deficits (Vora et al., 2013). A two-dimensional echocardiogram did not show any evidence of a patent ductus arteriosus or any other congenital defects (Vora et al., 2013).

Suspected carbon dioxide embolic events occurred shortly after insufflation

Cadis, Velasquez, Brauer, and Hoak (2014) conducted a case study on a 69-year-old female undergoing a laparoscopic cholecystectomy who suffered a suspected carbon dioxide embolism shortly after the trocar insertion for the creation of a pneumoperitoneum. The patient was placed in the supine position and the induction of anesthesia and endotracheal intubation occurred without complications (Cadis et al., 2014). Anesthesia was maintained with sevoflurane and the oxygen mixture and ventilator settings were not explicitly (Cadis et al., 2014). The surgeon placed the first 5 mm trocar under direct vision followed by the second trocar inserted into the umbilical region (Cadis et al., 2014). Immediately, the ETCO₂ decreased from 40 to 7 mmHg and shortly after to 0 mmHg (Cadis et al., 2014). The anesthesia provider immediately notified the surgeon, the pneumoperitoneum was released, and the patient was placed in left

lateral decubitus with reverse Trendelenburg (Cadis et al., 2014). Breath sounds were auscultated and the patient was hyperventilated with 100% oxygen with the lowest oxygen saturation reading 70% (Cadis et al., 2014). The blood pressure was 40/13 mmHg and promptly treated with 1 mg epinephrine and the blood pressure increased to 198/97 mmHg (Cadis et al., 2014). An arterial line was placed for more accurate blood pressure readings (Cadis et al., 2014). Nine minutes from the initial decrease in ETCO₂, the oxygen saturation increased to 100% and the ETCO₂ read 32 mmHg (Cadis et al., 2014). At this time, the arterial blood gas read: pH 7.244 with a PaCO₂ of 50.2 mmHg. The surgeon also observed bleeding at the trocar sites and decided to perform a laparotomy (Cadis et al., 2014). Once the abdomen was opened, the surgeon observed a small laceration in the anterior left lobe of the liver that did not have any active bleeding (Cadis et al., 2014). The patient remained stable throughout the remainder of the procedure and was extubated and transported to the recovery room (Cadis et al., 2014). Another arterial blood gas was drawn in recovery that showed a pH of 7.337 and PaCO₂ of 41.6 mmHg (Cadis et al., 2014).

Duncan (1992) describes a case study involving a 28-year-old female undergoing a laparoscopic tubal ligation that experienced a suspected carbon dioxide embolism. Endotracheal intubation and induction of anesthesia was uneventful and anesthesia was maintained with 70% nitrous oxide, 30% oxygen, and isoflurane between 0.9 and 1.5% (Duncan, 1992). Ventilation was controlled with a respiratory rate of nine and a tidal volume of 700 mL that produced a peak inspiratory pressure of 26 cm H₂O (Duncan, 1992). The patient was placed in dorsal lithotomy with a 45-degree Trendelenburg tilt (Duncan, 1992). A pneumoperitoneum was created with 2000 mL of carbon dioxide and the ETCO₂ was maintained between 32 and 34 mmHg (Duncan, 1992). Two minutes after insufflation, the ETCO₂ decreased to 16 mmHg and then quickly down to zero mmHg (Duncan, 1992). Isoflurane was discontinued, and the patient was hand-ventilated

at 100% oxygen (Duncan, 1992). Shortly after, ventricular tachycardia appeared on the ECG that was briefly precipitated by an irregular rhythm with PVCs (Duncan, 1992). A carotid pulse was palpable, blood pressure was unreadable, and 50 mg lidocaine was given intravenously (Duncan, 1992). After the lidocaine bolus, the heart rhythm converted to second-degree heart block.

Atropine 0.5mg was given, the blood pressure measurement read 63/40 mmHg, and the oxygen saturation was 71% (Duncan, 1992). However, ventricular tachycardia returned without a palpable pulse. Cardiopulmonary resuscitation was initiated, 1mg epinephrine given, and a dopamine infusion was started (Duncan, 1992). A cardiac defibrillator was not available in the operating room (Duncan, 1992). The patient's pupils were noted to be dilated, bilaterally (Duncan, 1992). About 5 minutes into cardiac compressions, a supraventricular tachycardia was noted on the ECG monitor with a palpable pulse (Duncan, 1992). The dopamine was discontinued because the heart rate was 160-200 beats per minute and the blood pressure was 220/100 mmHg. The pupils began to constrict and the patient started to spontaneously breathe and follow verbal commands approximately 30 minutes after the initial insufflation. At this time, the ECG showed sinus rhythm at 120-130 beats per minute (Duncan, 1992). About 60 minutes after the initial insufflation, the patient was extubated and sent to the recovery room and transferred to a cardiac stepdown unit (Duncan, 1992).

Gough and Thogulava (2014) conducted a case study involving a 33-year-old female undergoing a diagnostic laparoscopy for lower abdominal pain. A rapid sequence induction was performed without complication and anesthesia was maintained with sevoflurane, as well as, a nitrous oxide and oxygen mixture (Gough & Thogulava, 2014). Creation of a pneumoperitoneum was achieved through a Veress needle (Gough & Thogulava, 2014). Immediately following insufflation, Gough and Thogulava (2014) describe a decrease in ETCO₂ from 4.8 kilopascals

(kPa) to 1.0 kPa. In other words, the ETCO₂ decreased from roughly 36 to 8 mmHg. At the same time, the oxygen saturation decreased to 65%, the blood pressure was 48/25 mmHg, and the heart rate was 35 beats per minute (Gough & Thogulava, 2014). The ECG showed marked ST elevation in the anterior and inferior leads accompanied by periods of arrhythmias, including: ventricular tachycardia, supraventricular tachycardia, atrial fibrillation, and bigeminal and trigeminal rhythms (Gough & Thogulava, 2014). Interventions included: Trendelenburg position, 100% oxygen, release of insufflation, and rapidly intravenous fluid administration (Gough & Thogulava, 2014). Vital signs improved in two minutes from the initial drop in ETCO₂ and the ST elevations resolved within 15 minutes (Gough & Thogulava, 2014). Oxygen administration at 100% was continued, intravenous fluid administration was continued, and esmolol was administered to treat sinus tachycardia (Gough & Thogulava, 2014). Surgery was postponed and the patient was unsuccessfully extubated and required reintubation due to non-purposeful movement on the left side, lack of verbal response, and presence of agitation (Gough & Thogulava, 2014). A computed tomography (CT) scan and transthoracic echocardiogram was performed without any remarkable findings (Gough & Thogulava, 2014). An exploratory laparotomy was performed later that evening and involved removal of a ruptured ovarian cyst without complication (Gough & Thogulava, 2014). The following day, the patient was successfully extubated without any sign of neurological deficits (Gough & Thogulava, 2014). A second transthoracic echocardiogram was performed that showed a patent foramen ovale that was observed only during a Valsalva maneuver (Gough & Thogulava, 2014).

Kim, Park, Shin, Yi, and Kim (2010) describes a case study involving a 59-year-old woman with grave's disease undergoing an endoscopic total thyroidectomy using the axillo-bilateral-breast approach (Kim et al., 2010). The patient underwent intubation and induction of

anesthesia with sevoflurane and an oxygen and air mixture uneventfully (Kim et al., 2010). The ETCO₂ was kept within a range of 32 to 34 mmHg and the patient was placed in the supine position with neck extension accomplished with a shoulder pillow (Kim et al., 2010). Following the insertion of a 15 mm trocar into a neck incision with an insufflation pressure of 6 mmHg, there was a sudden decrease in ETCO₂ from 34 to 13 mmHg (Kim et al., 2010). At the same time, systolic blood pressure was 70 mmHg, oxygen saturation decreased from 99% to 88%, and the ECG monitor exhibited tall peaked T-wave changes with sinus rhythm and normal QRS complexes (Kim et al., 2010). The patient was quickly placed in the Durant's position, oxygen administered at 100%, and 5 mg ephedrine given (Kim et al., 2010). Carbon dioxide insufflation was stopped and an arterial blood gas measurement was obtained showing a PaCO₂ of 39.1 mmHg and PaO₂ of 84.5 on 100% oxygen (Kim et al., 2010). The vital signs returned to baseline about 5 minutes after the initial decrease in ETCO₂ (Kim et al., 2010). About 10 minutes after the start of the event, a TEE confirmed carbon dioxide embolism by exhibiting a "massive amount of gas bubbles in the right heart and a few gas bubbles in the left heart" (Kim et al., 2010, p. 775). There was no evidence of an intracardiac shunt (Kim et al., 2010). The procedure resumed without complication and the patient did not exhibit any cardiopulmonary or neurological deficits postoperatively (Kim et al., 2010).

Kondo et al. (2016) describes a case study involving a 69-year-old female undergoing a direct peroral cholangioscopy (DPOCS) to treat hepatolith using lithotripsy. The patient did not receive general anesthesia and was kept adequately sedated with midazolam (Kondo et al., 2016). The surgeon aspirated pus and mucus from the peripheral bile duct near the hepatolith prior to the initiation of lithotripsy (Kondo et al., 2016). As the hepatolith was fractured using a Holmium:YAG laser, "the patient suddenly went into shock and had a cardiac arrest", in which

the patient did not survive (Kondo et al., 2016, p. E215). A CT scan during cardiopulmonary resuscitation revealed multiple gas emboli in the systemic arteries and veins (Kondo et al., 2016). A post-mortem examination discovered intravascular gas and systemic gas emboli (Kondo et al., 2016). The official cause of death was systemic gas embolism and the authors believe that the aspiration of pus and mucus prior to lithotripsy may have opened a pre-existing biliary-venous shunt (Kondo et al., 2016).

Patel et al. (2012) describes a case study, in which, a 43-year-old woman undergoing laparoscopic lysis of adhesions experienced a carbon dioxide embolism confirmed by TEE. Her past medical history was notable of ventricular tachycardia requiring three cardiac ablations, and a recent transthoracic echocardiogram showed an ejection fraction of 55%, an estimated pulmonary artery systolic pressure of 36 mmHg, and moderate tricuspid regurgitation (Patel et al., 2012). Endotracheal intubation and induction was uneventful. After the creation of a pneumoperitoneum with a Veress needle, the patient's heart rate suddenly decreased from 60 to 30 beats per minute, accompanied by a reduction in oxygen saturation from 99% to 88% (Patel et al., 2012). The authors did not mention any change in ETCO₂ at this time. The pneumoperitoneum was released, the Veress needle was removed, and 0.8 mg of atropine was administered (Patel et al., 2012). Asystole occurred, ETCO₂ decreased, and chest compressions were initiated. Pulseless ventricular tachycardia developed and a biphasic defibrillator was used at 200 joules and the cardiac rhythm converted to ventricular fibrillation (Patel et al., 2012). Epinephrine 1 mg was administered and a second shock at 200 joules converted the ventricular fibrillation to sinus rhythm with a palpable pulse. Vital signs returned to stable condition, but steep and transient ST elevations occurred greater than 2 mm (Patel et al., 2012). A TEE was performed revealing gas in all found chambers of the heart, as well as, within the hepatic

circulation. The results of the TEE confirmed diagnosis of carbon dioxide embolism after “unintentional intrahepatic Veress needle placement” (Patel et al., 2012, p. e23). A patent foramen ovale was not found, left ventricular ejection fraction was 25% with global hypokinesis, reduced right ventricular function, and severe biatrial dilation (Patel et al., 2012). A transthoracic electrocardiogram performed three weeks later showed a left ventricular fraction of 60% and a patent foramen ovale was identified; however, a right-to-left shunt was not notable at rest (Patel et al., 2012).

Smith (2011) describes a case study involving a 34-year-old woman planned to have a dilation and curettage after a miscarriage. However, the surgeon requested to convert to an exploratory laparoscopy because of a concern that a surgical instrument may have been advanced too far into the abdomen (Smith, 2011). The patient was successfully intubated and the stomach was decompressed before trocar insertion (Smith, 2011). Sevoflurane was maintained at 2.3% with oxygen at 50% and air at 50% (Smith, 2011). About 3 minutes after insufflation, oxygen saturation decreased from 98% to 74%, heart rate dropped from 74 to 50 beats per minute, systolic blood pressure at 50 mmHg, and ETCO₂ at zero mmHg (Smith, 2011). Interventions included: 100% oxygen with manual ventilation, insufflation released, rapid intravenous fluid administration, five 10 mg ephedrine boluses given, and patient was repositioned to the Durant’s position (Smith, 2011). Approximately 2 minutes of resuscitative efforts, the patient’s systolic blood pressure returned 80 mmHg, heart rate 90 beats per minute, ETCO₂ 29 mmHg, and oxygen saturation of 98%. The surgeon insufflated the abdomen once more to reveal a uterine rupture. An emergent laparotomy was performed and repair of the uterine wall was successful. The patient lost a total of 2000 mL of blood requiring over 4 L of crystalloids, 3 units of pack red blood cells, and one L of Hespan (Smith, 2011). Methergine and Hemabate was also required to

help control the bleeding (Smith, 2011). The patient was extubated and recovered without any deficits and a postoperative hemoglobin and hematocrit was 10.4 g/dL and 31.8%, respectively (Smith, 2011).

Discussion of Case Studies

There are similarities and differences found between the case studies described above. With the exception of one case study, all the patients were female. The mean age was 36.4 years and the median age was 40 years. These statistics show that the majority of the case studies involved patients that are fairly young. Seven of the twelve case studies involved a carbon dioxide embolism that occurred directly after insufflation. Three of the twelve case studies involved performing a laparoscopic cholecystectomy. With the exception of one case study that involved a brief increase in ETCO₂ followed by a decreased in ETCO₂, all the cases involved a sudden decrease in ETCO₂. Seven of the twelve case studies involved significant ECG rhythm changes including: cardiac arrest, ST elevations, PVCs, and ventricular tachycardia. Only one case study discovered an audible mill-wheel murmur. One case study resulted in death caused by the presence of carbon dioxide emboli. Ten case studies specified the amount of time it took for the patient to return to hemodynamic stability and the average time was 9.3 minutes.

Case Study Limitations

The limitations to the case studies described above vary from case to case. Notable limitations to the diagnosis and treatment to the suspected carbon dioxide events described involve the lack of resources, level of expertise, and difference of opinion from one provider to another. In all the case studies, one major limitation is small sample size, which makes it extremely difficult to create recommendations for all carbon dioxide embolisms. In many of the case studies described, the use of a TEE was either not utilized or the provider did not have

access to one. The use of a TEE may help in the prompt diagnosis of a carbon dioxide embolism, but will not help treat carbon dioxide embolism. In one circumstance of cardiac arrest, a cardiac defibrillator was not available when needed and this may have contributed to a delay in treatment. Surgical technique played a role in all the case studies above, where carbon dioxide embolisms either occurred directly after insufflation or during critical surgical dissection. Another limitation that was found in numerous case studies, is the lack of a central venous catheter. In all, but one case study where a central venous catheter was in place, gas was able to be aspirated from the right side of the heart, possibly contributing to speedier recovery.

Personal Account of Suspected Carbon Dioxide Embolism

The patient was a sixty-eight-year old male undergoing a laparoscopic, hand-assisted left nephrectomy for a renal mass. General anesthesia was induced and intubation with an endotracheal tube was successful. After successful intubation, an arterial-line was placed and a second intravenous catheter inserted. Patient was then placed in right lateral decubitus with the bed flexed. General anesthesia was maintained with sevoflurane. Nitrous oxide was not used during the case. After induction, patient required a phenylephrine infusion for hypotension and eventually vasopressin boluses to maintain normal blood pressure readings. The patient had a continuous amount of blood loss resulting in a total of 2.2 L requiring two units of pack red blood cells, albumin infusions, and over 10 L of intravenous fluids. About 2.5 hours into the surgery, there was a sudden ETCO₂ drop from 40 to 18 mm Hg accompanied by a sudden decrease in arterial blood pressure to 60s/30s, and a reduction in oxygen saturation to 85%. Interventions included: telling surgeon to stop procedure, calling for help and the code cart, hand ventilating patient with ease, giving 50 mEq of sodium bicarbonate, epinephrine boluses, and starting patient on an epinephrine infusion. Patient was already receiving aggressive intravenous

fluids. Discussion of placing patient in supine position for the possible need for compression occurred, but patient responded to resuscitative efforts before that could occur. Patient's ETCO₂ and vital signs stabilized. A TEE was performed and the interpretation was that the ejection fraction was 65% with moderately hyperdynamic systolic function in the setting of a moderate dose epinephrine infusion, normal preload (patient was adequately fluid resuscitated), normal ventricular function, no evidence of valvulopathy, and no evidence of pulmonary embolism. The TEE was performed after the patient stabilized, which means the suspected carbon dioxide embolism must have dissolved into the bloodstream by then. The patient was transferred to the intensive care unit, the epinephrine infusion was stopped within a few hours of transfer, and the patient was extubated the next day without any signs of neurological deficits.

Unlike the most of the other case studies, this was male, older than the average age listed, experienced significant blood loss through the case, and had significant blood pressure instability requiring intervention before the event occurred. Through the research conducted in this paper, blood loss is known to be a risk factor for a carbon dioxide embolism and this patient clearly had a significant amount of blood loss before the event occurred. Another possible contributing factor in this case was that the tumor was slightly migrated into the renal vein. With discussions with the surgeons and anesthesia providers, it was concluded that surgical excision around the renal vein was most likely the culprit for the introduction of carbon dioxide into the pulmonary circulation.

Conclusion

Laparoscopic surgery is a common surgical approach. It is associated with less postoperative pain, faster recovery, and short hospital stays. While it has many advantages, providers must keep in mind that the rare complication of carbon dioxide emboli is possible.

Anesthesia providers play a crucial role in prompt recognition and treatment of carbon dioxide embolisms to provide life-saving measures to patients undergoing laparoscopic surgery.

Communication between the anesthesia provider and the surgeon is an important step providing prompt, efficient, and safe interventions to adequately resuscitate a patient suffering from a carbon dioxide embolism. If left unrecognized and untreated, carbon dioxide emboli can have devastating effects, including death.

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